SLG Best Practices - Examples

Introduction

This document accompanies the resource *Designing Quality SLGs: A Student-Informed Overview*. It is intended to provide concrete examples of how our recommended practices may be implemented in Self-Learning Guides (SLGs). The excerpts below are drawn from existing SLGs across the PISCEs curriculum and were selected because of how they exemplify each of the student-informed recommendations.

Each excerpt is not intended to prescribe a single best approach towards prework design. Instead, they are meant to offer initial clarity, inspiration, and reference, particularly for faculty who are new to SLG creation/revision or seeking clarification of items listed on the *SLG Best Practices* checklist. We invite faculty readers to feel empowered to adapt these principles in ways that best align with their teaching style.

Color Key

Excerpts from SLGs are contained within boxes.
Annotations are provided in dark red.

SLG is organized into logical heading and sub-headings

Excerpt from: Endo/Repro (2024) – Anterior Pituitary
Author: Sonia Ananthakrishnan MD

This is the content outline for the Anterior Pituitary SLG. All of the material contained within the SLG is organized under these headers and subheaders, each focusing on a particular background topic or function of the organ examined.

1. Anatomy of the pituitary gland
    a. Embryology
2. Hormones of the anterior pituitary
3. Approach to a mass of the pituitary
4. Pituitary hypofunction
    a. ACTH deficiency
    b. TSH deficiency
    c. Prolactin deficiency
    d. Gonadotropin deficiency
    e. GH deficiency
5. Pituitary hyperfunction
    a. Gene mutations
    b. Hyperprolactimenia
6. GH excess

The text of the SLG flows logically

Excerpt from: Neurology (2024) – Headache and Facial Pain
Author: Shuhan Zhu MD

The highlighted section connects the preceding introductory content to the remainder of the SLG. It adds a logical flow to the document, as the author provided framing and orientation regarding the order of the topics.

Now that you have a strong framework of how to evaluate patients for major secondary and primary disorders, please work through the rest of this syllabus (go to section titled Major Secondary Causes of HA not to miss) to review some major secondary headaches disorders NOT to miss such as:

- Giant Cell Arteritis (GCA)
- Idiopathic intracranial hypertension (IIH)

These are just a few secondary disorders that have headache as the most prominent component (early on), it is very important to use the SNOOP4 mnemonic to help alert you to these possibilities based on the details of the patient's headache. We have already reviewed SAH in the context of Neurological Emergencies (see the Neuro Emergencies SLG for additional details).

Text is broken up into easily digestible chunks

Excerpt from: Gastroenterology (2024) – Nausea and Vomiting
Author: Robert Lowe MD

Here, chunking in the form of creating small paragraphs increases readability of the text.

Treatment of Nausea and Vomiting
Therapy for nausea and vomiting is largely pharmacologic - restoration of fluid and electrolyte balance should be a priority, and several classes of pharmacotherapy are available to relieve symptoms.

Anticholinergic drugs – (e.g. scopolamine): particularly effective for motion sickness, as vestibular N/V are mediated at least in part through cholinergic pathways. Often provided in a transdermal delivery symptom (i.e. a "patch"). The use of anticholinergic medications is limited by the many side effects (dry, mouth, urinary retention, GERD).

Antihistamines (H1 blockers) – act centrally to reduce N/V. Agents such as meclizine are used primarily in vertiginous disorders, while diphenhydramine (Benadryl) has broader usage. The major side effect is drowsiness, which can limit the use of these agents.

Neuroleptic agents (e.g. prochlorperazine, chlorpromazine) – effective in GI illnesses, drug- and radiation-induced N/V. These drugs act by antagonizing cholinergic and histaminic stimuli, and by blocking dopamine D2 receptors in the CTZ. One advantage of these drugs is their availability in oral, injectable, and suppository forms. Side effects include sedation, dystonia, and tardive dyskinesia.

Key terms and concepts highlighted by bolding/changing text color

Excerpt from: Hematology (2023) – Overview of Hemostasis and Thrombosis: Primary and Secondary Hemostasis
Author: Caitlin Neri MD, MPH

Both bold and colored text are employed in this SLG for different purposes (organization vs. highlighting prototypical agents for learning).

You are only responsible for the prototype drugs and the mechanisms as described here. They are in **purple** in this SLG.
[…]
Antiplatelet Agents – There are a number of obvious therapeutic targets in the system to inhibit clot formation.
Aspirin – inhibits platelet aggregation by inactivating Thromboxane A2 via the enzyme cyclooxygenase
Clopidogrel – Inhibits platelet aggregation by binding to P2Y12 ADP receptors on platelets. This binding prevents activation of the glycoprotein GPIIb/IIIa complex and platelet aggregation.

Includes multiple formats (text, videos, figures, & tables)

Please see excerpts below for an example table and framing surrounding a video.

Video titles and length are listed in the overview

Excerpt from: Renal (2023) – Acute Kidney Injury
Author: Lauren Stern MD

This section is located at the beginning of the SLG and lists all required videos (and corresponding lengths) to orient students to the time needed to effectively progress through the document.
Required Videos:

AKI overview (8:31)
Pre renal AKI (20:04)
Intra renal AKI (24:09)
Post renal AKI (7:56)

Acute Kidney injury (AKI) is defined as the sudden decrease in kidney function over hours to days. A number of criteria including RIFLE, KDIGO, and AKIN have been developed and validated in various studies to help further define this syndrome. It should be noted that these criteria are most useful in a research setting where AKI has clearly defined parameters and can help the researchers to include and exclude subjects based on a more uniform criteria.

Video links are embedded throughout the SLG so students watch them in the appropriate sequence within the SLG material

Excerpt from: Cardiology (2023) – Bradyarrhythmias and Bundle Branch Blocks
Author: Omar Siddiqi MD

In this instance, helpful timestamps are added for student reference.

Watch Bradyarrhythmias and Bundle Branch Blocks (Watch 50:39-52:42) *[hyperlink].*

Left ventricular hypertrophy may be the result of many distinct processes. These include hypertension (LV pressure overload), genetic disorders (hypertrophic cardiomyopathy), infiltrative disorders.

There are many criteria for diagnosing LVH. Some of these criteria are as follows:
a. R in aVL > 11 mm
b. R in lead I > 15 mm
c. S in V1 + R in V5 or V6 ≥ 35 mm
d. Cornell criteria: S in V3 + R in aVL > 28 mm (men) and > 20 mm (women)

Use bullet points/lists to summarize key concepts from the videos

Excerpt from: Foundations 1 (2023) – Proteins in Health and Disease
Authors: Karen Symes PhD, David Harris PhD

Please watch the following video on the role of Hsp70 in protein folding *[hyperlink].*

Key points from the video:

- Levels of Hsp70 are increased due to cellular stress
- Hsp70 mechanisms:
    - Repairs damaged proteins and corrects misfolding
    - Prevents protein aggregates and facilitates removal of protein aggregates
    - Promotes lysosomal function

Limit amount of background material presented to relevant info

***Excerpt from*:** Foundations 3 (2023) – Humoral Immune Responses
Authors: Daniel Remick MD, Rithik Reddy

The background information of this SLG is tightly compressed into the highlighted paragraph.

1. **Big picture:** Your body produces antibodies to protect you from infections. Complement proteins also help eradicate bacteria. Vaccines induce antibodies to protect against infections.

2. **Introduction:** The humoral immune response is a key mechanism that targets extracellular microbes and microbial toxins. Antibody production by mature B cells is a protective, adaptive mechanism that allows responses to the large diversity of pathogens the body might encounter. The complement system is composed of a cascade of proteins that primarily eradicate bacteria.

3. **Initiating the response:** Initially, a naïve B cell recognizes an antigen that activates the B cell, triggering proliferation and differentiation into a variety of B cell subtypes that combat the pathogen (Fig 7.1). T cells require antigen presentation on MHC molecules but B cells can recognize free antigens.

4. **Further B cell responses:** Naïve B cells, initially activated by recognizing an antigen, become further activated by helper T cells (CD4+ cells) that stimulate both increased affinity and increased numbers of memory B cells. These B cells provide specialization and longer-lasting antibody responses.

Use lists or tables to present and summarize complex text

Excerpt from: *Foundations 3 (2023) – Antifungal Agents*
Author: Daniel Borque MD

The introduction of this SLG contains the table below, which efficiently identifies all of the pharmacological classes and agents that students are responsible for learning, creating a helpful reference point.

Antifungal Class	**Prototype**
Polyene	amphotericin B
Azoles	fluconazole
Echinocandins	micafungin
Flucytosine	flucytosine